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You are working one evening, and the EMS dispatch center calls. The ambulance is bringing in a 35-year-old male motor vehicle collision victim who is unresponsive and has a BP of 80 palpable. So, what happens next in your emergency department? Who do you assemble? What equipment do you gather? Do you call the blood bank and the operating room?

Many of you have heard the American College of Surgeons mantra that trauma is a surgical disease. Like all aphorisms, there is an element of exaggeration in this statement. While some trauma victims may require surgery, many, if not most, trauma victims do not need surgery in the operating room. I would comment that trauma is becoming more than just a surgical disease, as evidenced by the skill with which surgeons are non-operatively managing more and more trauma victims. As non-operative management becomes more common, appropriate assessment, often with resuscitation in the emergency department, is increasingly important. Therefore, assessment and resuscitation of traumatic shock should be part of an emergency physician's skill set.

—J. Stephan Stapczynski, MD, FACEP, FAAEM, Editor

Approaching Shock in the Trauma Patient

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Introduction and Epidemiology

In 1872, S. D. Gross described shock as, "a rude unhinging of the machinery of life."[1] He correctly viewed shock as a malfunction of cellular metabolism. A more contemporary definition is that of inadequate tissue perfusion. Although we may know that shock exists, knowledge of its cause must guide efforts for resolution. In trauma patients, shock is thought to be of hemorrhagic etiology until proven otherwise, largely because this etiology of shock is the most time-sensitive to correct. We have come to learn that the consequences of shock may be mitigated by early recognition and intervention.

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The challenge facing providers (particularly those first to provide definitive care) is that of approaching shock with a plan and maintaining the ability to react quickly when that plan is modified by deteriorating physiology. This article will review general considerations for shock and trauma care, as well as injury-specific diagnosis and management.

Often underappreciated by the public, trauma is the leading cause of death in people ages 1-44 and the fourth "overall" cause of death in the United States.[2] More than 150,000 Americans die each year as a result of unintentional or intentional injury.[3] Motor vehicle trauma is the leading cause of injury-related death in the United States, and rural settings account for 42% more fatal crashes than do urban settings. Among deaths occurring within one hour of hospital admission, 47% were motor-vehicle related. Gunshot wounds were next, at 31%.[4] Trauma patients present in various stages of physiologic dysfunction and dysregulation, and 5-15% of trauma admissions are considered to be in shock on arrival to the emergency department.

The Golden Hour

The "golden hour of trauma" is the 60 minutes following traumatic injury, and is believed to be the most critical time period for intervention to halt the physiologic demise of a patient in shock. This critical intervention begins in the field with first aid from bystanders and advanced care from EMS and only then proceeds to the emergency department, operating room, and intensive care unit. The concept of the "golden hour" has been described to extend through the "silver day," as the extremes of the physiologic response to trauma are triggered very early and can be prolonged.[5] Rapid intervention remains the mainstay of therapy to avoid later multiple organ dysfunction. This concept has changed our initial resuscitative strategies in several areas. One area of change is the question of the endpoint goal for pre-hospital care in trauma. To what extent should pre-hospital providers "scoop and run"? To what extent is it appropriate for them to "stay and play"?[6] Which patients should go to a trauma center?[7] Because emergency physicians are often the "medical control physicians" for EMS agencies and helicopter programs, it is of great value to provide input to these agencies when their protocols are being developed. Berlot[8] describes that the pre-hospital providers have two major goals, including securing the airways and restoring tissue perfusion. No single approach is appropriate in all settings. For example, some EMS agencies have endorsed rapid sequence intubation, surgical airways, and central venous access in appropriate settings, while other agencies provide standard basic life support care. The emergency physician should be aware of the level of pre-hospital care in his or her area, as well as enable these technicians to adequately intervene in the "golden hour."

The Role of the Assembled Trauma Team

Another major responsibility of emergency physicians and nurses is to make or contribute to hospital management plans for trauma patient arrivals. A critical concept is the extent to which surgeons should have early involvement in trauma management decisions. Prior to the evolution of assembled trauma teams, emergency physicians often provided excellent and timely initial evaluations, summoning surgeons only when it was felt that a patient required operation. Other thinking in this regard suggests that surgeons should be summoned even before patient arrival, as based on pre-hospital information.

The literature in emergency medicine increasingly suggests that non-surgeons should be involved as trauma team leaders.[9,10] The American College of Surgeons views trauma as a surgical disease and suggests that trauma surgeons should be present immediately when certain types of patients arrive.[11] There is certainly a plethora of polarized literature on this concept. Perhaps a more appropriate way to think about this concept is that trauma designation and verification presently mandate that for certain injuries, a surgeon must be included immediately in the multidisciplinary team. The ability of emergency physicians and surgeons collectively to embrace this concept often is a critical determinant in trauma center verification or designation and tends to allow a more collaborative approach.

It is critical that trauma team members understand their roles in the initial management of the severely injured. The arrival of the patient to the resuscitation area is the wrong time to determine which provider performs which tasks. Increasingly, there is a role for formal team training to be done without a patient and

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Table 1. Common Indicators of Shock

PHYSICAL FINDINGS	
	• Decreased LOC/combativeness/stupor
	• Cool, "clammy" skin
	• Diminished pulses
	• Capillary refill > 2 seconds
	• Poor urine output
OBJECTIVE VITAL SIGNS	
	• Tachycardia
	• Hypotension
	• Tachypnea
	• Hypothermia
METABOLIC MARKERS	
	• Elevated base deficit
	• Decreased pH
	• Elevated lactic acid
	• Low bicarbonate

in simulation scenarios.[12,13] The trauma bay is a place for designation of duties, designation of authority, and designation of responsibility. In this arena, duty, authority, and responsibility are not synonymous at all. It is a place for symphonic, collaborative communication. Yelling and shouting must be avoided because they impede effective communication. Further, yelling gives an impression of chaos to pre-hospital providers.

Trauma Team Activation

Once it is known that an injury has occurred, a reasonable (but not certain) assumption is that if shock is present, it is the result of the injury. Exceptions include cardiogenic or septic shock that may have preceded a minor traumatic event. An important consideration is how to respond to this knowledge. The American College of Surgeons endorses the idea that trauma team activation criteria should be established to allow clinicians to respond promptly to pre-hospital information.[14] These criteria are commonly physiologic for the highest level of activation and situational for the second tier of activation. Hospitals with experience and excellence in trauma care often are designated by a regulatory authority or verified by the American College of Surgeons (ACS) as a trauma center of varying levels. These institutions have learned by experience that mobilizing the correct individuals prior to the arrival of the patient (when notified early) is the best approach to care. Commonly, the highest level of activation prompts a surgeon to be present with the emergency physician within 15 minutes of patient arrival. The minimum requirements from the ACS for the highest level of activation include:

• confirmed systolic blood pressure < 90 at any time in adults and age-specific hypotension in children;

• gunshot wounds to the neck, chest, or abdomen;

• GCS < 8 with mechanism attributed to trauma;

• transfer patients from other hospitals receiving blood to maintain vital signs;

• intubated patients transferred from the scene, or patients with respiratory compromise or obstruction (excludes patients intubated at another facility and now stable from respiratory standpoint);

• emergency physician discretion.

Second-tier (commonly referred to as Level 2) team activation criteria are less clear and often are chosen by the hospital. The key is to choose highly predictive variables in a simple fashion.[14] The emergency physician or designee should listen to the pre-hospital report and have some sense of understanding as to whether assembly of a trauma team would benefit the patient. This clinical judgment remains critical to timely care and patient safety. The emergency physician must listen for phrases or situations that increase his or her suspicion for significant injury. Often, these second-tier or Level 2 criteria are best chosen locally, based on the hospital's understanding of both overtriage and undertriage.[15] Although the triage recommendations for the highest level of activation have been challenged, it is fairly widely recognized that they allow the early involvement and input from a surgeon, and that this input is likely to be beneficial to the emergency physician.[16]

Overtriage and Undertriage

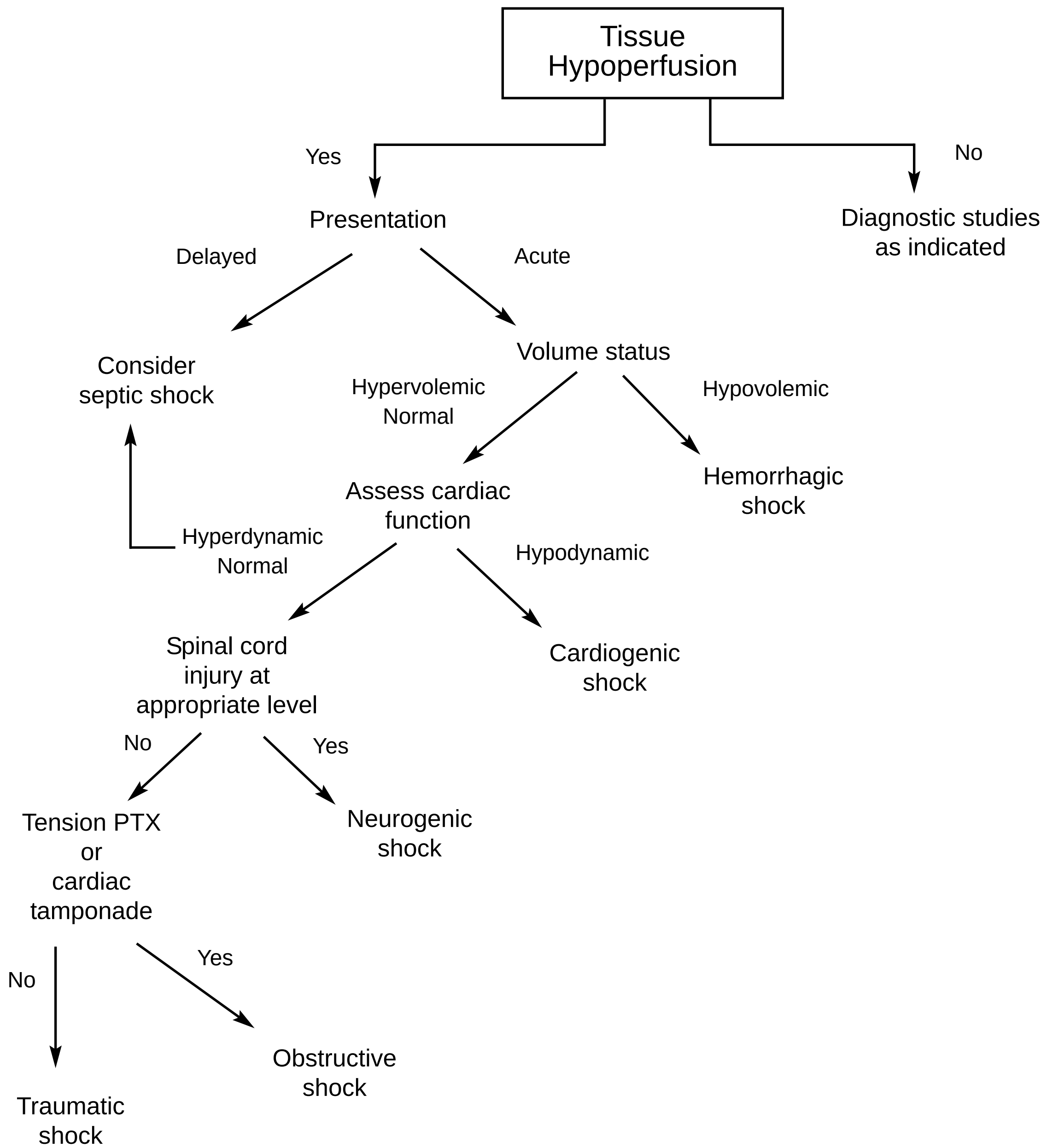
Overtriage is generally accepted to represent excessive resource allocation and is thought to result in excessive cost. Undertriage, as it pertains to trauma, similarly represents insufficient initial allocation of resources and may result in both delay of diagnosis and patient risk. Cribari[17] and others are studying the extent to which overtriage is expensive and the extent to which undertriage is dangerous. Regardless, an understanding of the relationship of mechanism as it pertains to common injuries is a key to the maturity of a hospital's trauma efforts. Ciesla[18] has described that excessive overtriage may be the result of immaturity of a trauma system and further describes that this circumstance should result in both refinement of local criteria and development of a regional system.

Common Considerations for the Trauma Patient in Shock

The approach to the trauma patient in shock must be a systematic process with an initial primary survey, resuscitation of vital functions, a detailed secondary survey, and only then, injury-specific definitive intervention.[19] Shock in the trauma patient is identified during the primary survey and is managed simultaneously as the rest of the primary survey is completed.[20] Certain mechanisms often are associated with specific injury. For example, left-sided blunt abdominal trauma often results in splenic injury. Burns of a child's feet and ankles are often suggestive of immersion and child abuse.[21] Pedestrians struck by automobiles often suffer lower extremity fractures and neurotrauma. Clinicians must use and interpret a patient's mechanism of injury to assist in the diagnosis and management of both obvious and easily missed injuries.

Shock at its most basic level is simply, "inadequate tissue perfusion." It is important for the trauma team to understand that the

Figure 1. Tissue Hypoperfusion Algorithm



Reprinted with permission from: Moore, et al. *Trauma*, 5th ed. New York: McGraw-Hill; 2003: figure 13-5, page 222.

diagnosis of shock does not hinge on any single piece of information. A patient who is normotensive may indeed be in shock. Actually, it has been shown that systemic hypotension is a late marker of shock after traumatic injury. A significant degree of "compensated" shock may be present long before the systolic blood pressure decreases to the widely accepted 90 mmHg systolic blood pressure (SBP) that historically equals shock.[22] A trauma patient's initial SBP correlates poorly with arterial base deficit, a marker of inadequate end-organ perfusion. It was found that mean and median SBP did not decrease to less than 90 mm Hg until the base deficit approached -20, with an associated mortality of 65%.[23] This illustrates the compensatory ability of the body to maintain blood pressure for vital organ perfusion while causing ischemia to the other organ systems.[24]

Identification of shock can be achieved through physical examination, objective vital signs, and metabolic markers. In early compensated shock, the diagnosis often is difficult. Multiple findings may indicate tissue hypoperfusion.[25] (*See Table 1.*)

Once shock has been identified, the goal of the trauma team is to stop the insult as well as to resuscitate the physiologic losses of the patient. The exact cause of shock should be determined. The differential diagnosis of shock in the trauma patient includes hemorrhagic/hypovolemic, obstructive, cardiogenic, neurogenic, and distributive/septic shock. The trauma patient in

Table 2. Classes of Hemorrhage (70 kg Patient)

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood Loss (cc)	0-750	750-1500	1500-2000	> 2000
% of Blood Vol	0-15%	15-30%	30-40%	> 40%
Heart Rate	60-100	100-120	> 120	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Mental Status	Anxious	Anxious	Anxious/confused	Confused/lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid/blood	Crystalloid/blood

shock is considered to be in hemorrhagic shock until proven otherwise, as this is by far the most common type of shock encountered. (*See Figure 1.*)

All trauma patients should be assessed in the standardized fashion above, preferably in a warm, dedicated resuscitation area with supplies/adjuncts immediately available to treat shock. Airway control with supplemental oxygenation is required, as well as large-bore intravenous access in all trauma patients at risk for shock. Rapid availability of radiology personnel and equipment is necessary. Even very small centers can evaluate and stabilize trauma patients safely with a well coordinated trauma team that knows what capabilities it does and does not have on hand.

Hemorrhagic/Hypovolemic Shock

Hemorrhage is the most common cause of shock and leading cause of death in the trauma patient, and virtually all multiple-injury patients have an element of hypovolemia.[26] Penetrating and blunt force injuries can cause very obvious hemorrhage as well as less conspicuous bleeding. Hemorrhage-induced hypotension in a trauma patient is predictive of a mortality greater than 50%.[26,27] Hemorrhage and hypovolemia decrease intravascular volume and therefore reduce cardiac filling pressures, according to Starling's Law. As filling pressures decrease, the heart must increase its contractile rate to maintain cardiac output and systemic perfusion, and tachycardia is observed clinically. Hemorrhage also causes a loss of oxygen carrying red cells and important clotting factors.[28] The American College of Surgeons and its Advanced Trauma Life Support guidelines divide hemorrhage into 4 classes. Table 2 illustrates the progressive physiological response, clinical findings, and fluid requirements of the different classes of hemorrhagic shock. It is important to remember that individual physiological reserve varies among trauma patients, as well as the ability to mount a compensatory response to hemorrhagic shock. Advanced age, pregnancy, preexisting medical conditions, medications, and many other factors can alter a patient's response to shock.

The common sites of traumatic hemorrhage are external, intra-thoracic, intra-abdominal, pelvic/retroperitoneal, and extremity fractures with associated hematoma. All of these sites can be subject to both penetrating or blunt force and sometimes a combination of the two. Table 3 illustrates the volume of blood loss that can occupy injured body compartments.

Identification of Hemorrhage

The EMS report can help provide the information needed to quantify the amount of external hemorrhage in the field. Patton et al, demonstrated that quantifying external blood loss is difficult, by finding in an experimental setting that only 24% of EMS blood loss volume estimates are within 50% of the actual lost volume.[29] Extensive external hemorrhage may occur from even small lacerations in vascular areas such as the scalp and perineum and should be quickly controlled.

Physical examination coupled with chest x-ray, pelvic x-ray and bedside "FAST" (Focused Assessment with Sonography for Trauma) allows identification of the site of significant internal hemorrhage in most cases. Examination is directed to rule out hemorrhage in each of the above-described bodily compartments. An anterior-posterior chest film allows evaluation for mediastinal enlargement, hemothorax, and pneumothorax. Controversy surrounds routine chest x-ray on stable trauma patients; however, all patients with shock-like presentations should have a chest film during the initial resuscitation.[30,31] Pelvic x-rays should be obtained in patients with physical examination findings of pelvic trauma and high-impact mechanisms of injury. Fracture of the pelvis can be associated with life-threatening retroperitoneal hemorrhage. FAST is an accepted screening tool for identifying hemoperitoneum and hemopericardium in the trauma patient, with a sensitivity reaching 95% when performed and interpreted by a practitioner formally trained in its use.[32] The hemodynamically unstable trauma patient with abdominal "free fluid" found on FAST examination should proceed directly to the operating room for laparotomy.[33,34]

Management of Hemorrhage

Applying direct pressure over a bleeding wound is a simple maneuver that often is overlooked. All but the most complex vascular injuries can be controlled in this manner. In extremity trauma, proximal pressure points can be compressed (femoral artery, brachial artery) to stop distal hemorrhage if direct pressure does not result in hemostasis. If extremity hemorrhage cannot be controlled via direct pressure, an arterial tourniquet such as a blood pressure cuff, Spanish windlass, or manufactured tourniquet can be applied just proximal to the wound. Arterial tourniquet times of up to two hours have been used safely.[35] "Hemostat" or surgical clamp placement is best reserved for the

Table 3. Sites of Common Blood Loss with Associated Volumes

SITE	BLOOD LOSS (CC)
Thorax	> 5,000
Abdomen	> 3,000
Pelvis	> 2,000
Femur fracture	1,000-1,500
Humerus fracture	200-500
Tibia/fibula fracture	400-700

Table 4. Intravenous Access Generalizations

- Peripheral access first
- Use the uninjured upper extremity first
- Use short, large-diameter catheters
- Use large-diameter drip set (blood tubing)
- Advanced access (central, "cut-down," intraosseous, etc.) is placed as the clinical circumstance and physician familiarity dictate
- Pressure infusing equipment should be immediately available
- First lab sent: Type and cross-match

operating room. Significant hemorrhage from the scalp can occur and must be controlled immediately with suture, staples, or "Raney" clips. Hemodynamically unstable patients with suspected pelvic fracture should undergo immediate external pelvic compression or fixation. Several modalities, including bed sheet wraps, MAST trousers, pelvic binder, and mechanical external fixators, can accomplish the goal of pelvic fracture stabilization with reduction of pelvic volume. Trauma patients who are found to have evidence of internal or uncontrolled hemorrhage need immediate availability of definitive surgical care (facilities, staff, and surgeon).

Hemorrhagic shock patients should be categorized as resuscitation responders, transient responders, and non-responders. Patients who initially have signs of shock but respond to initial IV fluid therapy and maintain adequate tissue perfusion are considered responders. Transient responders initially respond to resuscitative efforts with subsequent hemodynamic deterioration, and often signify a subset of patients with active, ongoing hemorrhage. These transient responders require emergent intervention (surgery, angioembolization, etc.). Patients who do not respond to any resuscitative therapies despite adequate control of hemorrhage are considered non-responders and most likely have suffered severe physiologic insult and possibly have irreversible shock.[36] A lethal triad of hypothermia, coagulopathy, and acidosis perpetuates in decompensated hemorrhagic shock, and aggressive treatment is required to halt this bloody vicious cycle.

Volume Resuscitation

Resuscitative fluid therapy is best initiated with peripheral venous access. Two large-bore peripheral IV catheters (16 gauge or larger in the adult) should be placed in all trauma patients at risk for shock. Central venous access sometimes is necessitated when multiple peripheral access attempts have failed. Large-bore central venous cannulas, coupled with a rapid infuser/warmer, allow for massive transfusions in minutes and should be readily available. During IV insertion, baseline laboratory studies should be sent, including: type and cross, hematocrit, and pregnancy test for all females of child-bearing age.[37] (*See Table 4.*)

Intraosseous access has long been a secondary option for fluid resuscitation in children. Experience has demonstrated that this approach may be a useful option in adults. Manual placement of standard intraosseous needles historically has been technically difficult in adults due to their dense bone cortex. The development of mechanical or powered tools to place intraosseous needles now allows this approach to be used in adult trauma victims. These adjuncts allow placement in as quickly as 10 seconds of a 15-gauge needle into the proximal humerus or tibia, which can achieve fluid administration flow rates of 60-100 cc per minute. Also, if marrow blood can be aspirated, it can be used for most laboratory tests, including transfusion cross-matching.

The choice of IV fluid used for trauma resuscitation has been the source of great debate for decades. The "crystalloid vs. colloid" debate is beyond the scope of this article, but there has yet to be a study that shows a distinct advantage of colloid/hypertonic solutions over isotonic crystalloid solutions (lactated Ringers or normal saline). The Advanced Trauma Life Support Course recommends the infusion of two liters of isotonic crystalloid solution to adult trauma patients presenting with shock.[20] This is based on a tachycardic 70 kg patient (5-6 liters of circulating blood volume, with probable 15-30% blood loss). The initial two liters of crystalloid should be followed with blood products if the patient's hemodynamic instability does not improve (non-responder) or deteriorates after initial improvement (transient responder). Blood product and crystalloid resuscitation should continue until hemodynamic stability is obtained in the transient responders and non-responders. Type O negative blood should be transfused only until cross-matched blood is available. It is important to remember that only 11-33% of infused isotonic crystalloid solution remains intravascular at one hour post infusion.[38]

Hypertonic crystalloid resuscitation for traumatic shock patients currently is being investigated for several reported advantages. Smaller transfusion volumes of hypertonic crystalloid can produce greater intravascular volumes than can be achieved with traditional isotonic resuscitation. Hypertonic solutions also have been reported to help suppress neutrophil "oxidative burst" activity and therefore possibly attenuate the body's immunological response to shock.[39,40] Currently there are ongoing NIH studies attempting to better understand this potentially advantageous resuscitation modality.

Massive Transfusion

Massive transfusion is defined by most as the transfusion of 10 or more units of packed red blood cells (PRBCs) within 24 hours.[41] The need for massive transfusion in the civilian setting occurs in only 1-3% of trauma patients and is associated with high mortality. Extensive blood bank resources are necessary to keep up with massive transfusions in acute settings. Blood product transfusion in the past has not been largely protocol-driven, and often transfusion ratios of PRBCs to thawed plasma and platelets vary from institution to institution.

Recently, as a product of military medicine in Iraq and Afghanistan, there has been landmark research on blood product transfusion in the hemorrhagic shock patient.

A number of large studies have shown that transfusing PRBCs in a 1:1 ratio with thawed plasma, as well as the early transfusion of platelets (before 10 units of PRBCs), is beneficial to these severely injured patients.[41-43] It makes logical sense to transfuse blood products in ratios that approximate whole blood, as a bleeding patient not only loses the oxygen-carrying PRBCs, but also the clotting factors in plasma. Physicians and nurses should be aware of their own facility's blood bank capabilities, as they vary greatly from institution to institution. Patients requiring massive transfusions also should be warmed actively, as hypothermia is common in these patients. Massive transfusion is associated with a multitude of complications and, therefore, goal-directed therapy to reach endpoints of resuscitation (discussed later) should be used to avoid over-resuscitation. Immunologic suppression, transfusion reaction, dilutional coagulopathy, and ARDS, as well as volume-associated complications such as abdominal compartment syndrome are common complications of massive transfusion.[41]

Currently there is much interest in the role of recombinant activated factor VII in the trauma patient with severe hemorrhage. While the medication has FDA approval for hemophilia patients undergoing surgery, several studies have looked at the off-label use in the trauma population. The results have proved promising, especially in coagulopathic patients and those with intracranial hemorrhage.[44,45] Future studies will investigate optimal indications, dosing, and potential complications of factor VII administration in the trauma patient population.

Permissive Hypotension

Another controversy in the pre-hospital and emergency department setting is that of the degree of volume resuscitation. There is tremendous ongoing work in this area. Mattox et al[46] and others have facilitated the phrases "permissive hypotension" and "hypovolemic resuscitation." It has been shown that large volumes of crystalloid resuscitation, especially for penetrating torso trauma, result in worse outcomes.[47] Some feel that if large volumes of crystalloid are administered to patients in shock, the iatrogenically elevated blood pressure may "pop the clot" or disturb the body's ability to form thrombus, as well as increase the rate of hemorrhage. The permissive hypotension strategy advocates withholding crystalloid resuscitation if there is other evidence of tissue perfusion (palpable pulses, mentation, or urine output). Many believe hypotension (after traumatic injury) to be an innate protective mechanism against death. This may prove to be one of the most significant controversies in medicine in the 21st century. The emergency physician and emergency nurse should be aware of this work and should attempt to come to some institutional consensus in their hospital as to whether hypovolemic resuscitation strategies are appropriate for penetrating torso trauma, blunt torso trauma, or for all forms of trauma. Ideally, this institutional consensus should expand to include the hospital's EMS agencies and sending hospitals for transfer patients.

Emergency Department Thoracotomy

Emergency department thoracotomy (EDT) can be used in an attempt to salvage critically injured patients who present in extremis. EDT can be considered in centers with immediate access to a surgeon and an operating room/staff. Successful endotracheal intubation, mechanical ventilation, and vascular access are mandatory in patients considered for EDT. Although there is some controversy regarding who should receive EDT, several guidelines exist.[48,49]

Patients who present with penetrating thoracic trauma, who have signs of life in the field, and have had less than 15 minutes of CPR are ideal candidates for EDT.

Patients who present with non-thoracic penetrating trauma, who have signs of life in the field, and have had less than 5 minutes of pre-hospital CPR are candidates for EDT.

In general, victims of blunt trauma are candidates for EDT only if they present with less than 5 minutes of pre-hospital CPR and have pulseless electrical activity (not asystole) as their presenting heart rhythm.

Once the left chest is opened in EDT, several potential life-saving maneuvers can be performed including: open cardiac massage, aortic cross-clamping, evacuation of pericardial tamponade, and direct control of intra-thoracic hemorrhage.

Obstructive Shock

Obstructive shock is used to describe the inadequate perfusion that accompanies these conditions. Two examples, cardiac tamponade and tension pneumothorax, frequently are observed in the trauma patient population and are a result of both penetrating and blunt trauma. In both conditions, venous return to the heart is obstructed by compression of the vena cava and right atrium/ventricle, as these are low-pressure systems.

Cardiac Tamponade. Traumatic cardiac tamponade is the result of blood accumulating between the pericardium and the heart itself and can be the result of both penetrating and blunt trauma. As little as 50-75 cc of blood can produce the clinical picture of critical cardiac tamponade.[50,51] Tamponade classically is associated with Beck's triad of distended neck veins, distant heart sounds, and hypotension.[52] Chest x-ray may reveal an enlarged heart shadow. The FAST examination has a reported 95-99% sensitivity in detecting tamponade in penetrating trauma, so the cardiac view always should be part of the procedure.[53] The sensitivity of the FAST examination detecting tamponade does decrease significantly in the setting of hemothorax, so caution must be used in interpreting the sonography in these patients.[54] Cardiac tamponade also must be aggressively ruled out in patients with hypotension but without hypovolemia or tension pneumothorax.

Once the diagnosis of cardiac tamponade is made, aggressive fluid resuscitation should be continued to increase venous return and augment the heart's preload (filling pressure). Evacuation of the pericardium has been described with needle pericardiocentesis, sub-xiphoid pericardial window, and thoracotomy. The decision of which modality to use depends on the patient's overall condition and the resources available at a given facility. Regardless, removal of as little as 20 ccs of blood may improve the hemodynamics of a patient in true cardiac tamponade.[55] Temporizing measures must be followed with prompt definitive surgical care.

Tension Pneumothorax. Pneumothorax is the term used for the accumulation of air between the chest wall and the lung itself, usually as a result of an injury to the bronchial tree or lung. Both blunt and penetrating trauma can cause pneumothorax. If the accumulation of air in the pleural space reaches a critical volume, both ventilation and venous return to the heart are impaired, causing a tension pneuomthorax and intractable shock. Tension pneumothorax classically presents with decreased breath sounds on the side of the pneumothorax, hyperresonance to percussion, tracheal deviation to the opposite side, hypotension, and tachycardia.[56]

A patient with shock-like symptoms and the classic physical examination findings of tension pneumothorax should undergo emergent needle decompression or immediate tube thoracostomy placement prior to radiographic confirmation. If the patient is treated initially with needle decompression for tension pneumothorax, tube thoracostomy still is required to treat the remaining simple pneumothorax. Currently there is controversy surrounding the management of the "occult" pneumothoraces, which routinely are found on chest CT scan and not on the standard AP plain film. It now is thought that greater than 70% of all traumatic pneumothoraces are occult.[57] In non-ventilated patients with simple pneumothorax, it now is acceptable to withhold thoracostomy with close observation.[58-60] However, patients with occult simple pneumothoraces who require positive pressure ventilation should have chest tube placement because up to 25% will have further lung collapse with the possibility of tension pneumothorax and shock.[61]

Cardiogenic Shock

Cardiogenic shock refers to the lack of systemic perfusion seen in cardiac failure. Cardiogenic shock is encountered in the trauma patient population, and although it is much less common than hemorrhagic shock, it is increasing as more elderly people are becoming victims of trauma. It can be a direct result of blunt or penetrating cardiac trauma that injures the myocardium or valvular structures. Patients with blunt thoracic trauma are subject to a variety of cardiac injuries, from minor myocardial contusion to complete cardiac rupture. Blunt cardiac injury is thought to be present in severe thoracic trauma up to 70% of the time.[62] Pre-existing comorbid cardiac disease will continue to be present in a significant number of trauma patients as the population ages. Motor vehicle collisions, falls, and other trauma often are the result of an acute cardiac event. A high index of suspicion is needed to evaluate for a cardiac event in a patient with shock and a relatively minor mechanism of injury, once hemorrhagic shock has been excluded. Decreased cardiac output, whether from an acute injury, acute coronary event, or chronic failure, is exacerbated in all types of shock. As the sympathetic system attempts to increase cardiac output for systemic perfusion, there is increased myocardial oxygen consumption, and therefore more strain on the already failing heart.

Identifying cardiogenic shock in the trauma patient requires a thorough review of the patient's medical history and the use of several diagnostic modalities. During the exposure of the patient, scars on the chest or an AICD/pacemaker should be noted and should alert staff of a potential cardiac history. A 12-lead electrocardiogram should be obtained.[63] There are no specific ECG findings associated with blunt cardiac injury, and nonspecific changes such as tachycardia or conduction delays are present in up to 80% in patients with blunt cardiac injury (BCI).[64] Although most BCIs involve the right side of the heart, the use of right-sided precordial leads has not proven to aid in the diagnosis of BCI.[65] A normal ECG has a negative predictive value of 90% for identifying cardiac injuries that require treatment.[66] Although the majority of ECG findings related to BCI are observed on admission, patients with mechanism for BCI and who are older than 55 years of age or have history of cardiac disease should have a 24-hour period of observation in a telemetry unit.[63] The routine use of cardiac enzymes for diagnosis of traumatic cardiac injury is controversial. Wisner[67] reported a review of 2252 blunt trauma patients with a normal ECG and found that none required cardiac-specific treatment. However, it generally is agreed that an abnormal admission ECG attributed to BCI mandates cardiac enzymes. Echocardiogram does not seem to have value in the screening of hemodynamically stable patients suspected of having BCI; however, echocardiogram is vital in cardiogenic shock to aid in the diagnosis of contusion or valve injury.[68]

The management of traumatic cardiogenic shock in the emergency department is centered around the standard protocols of the American Heart Association's Advanced Cardiac Life Support program. Secondary insult to the heart can occur with hypovolemia and anemia, and any hemorrhagic component of shock must be treated promptly. Inotropic support may improve cardiac output in the volume resuscitated cardiogenic shock patient.[69] Invasive monitoring, including pulmonary artery catheterization, may be warranted to direct volume and pressor support in patients with cardiac injury, especially in elderly patients with known cardiac disease and abnormal admission ECG.[70] Prompt consultation with cardiology or cardiothoracic surgery may be required in patients with acute infarctions, cardiac ruptures, and valvular dysfunction.[71] Intra-aortic balloon pump therapy has been used successfully in the patient requiring hemodynamic support in severe cardiac contusion.[72]

Neurogenic Shock

When the spinal cord is disrupted or injured from the cervical to mid-thoracic levels, sympathetic tone to the heart and peripheral vasculature may be impaired. There is resultant peripheral dilation and inappropriate bradycardia that create the condition neurogenic shock. As with all other trauma patients presenting with shock-like symptoms, hemorrhagic shock must be excluded first, even in patients with obvious vertebral or spinal injury. The presence of bradycardia does not necessarily equal neurogenic shock, as beta-blockers, BCI, or pre-existing conditions may cause this finding. Most often, treatment for neurogenic shock must be initiated before a specific spinal injury is diagnosed.

Along with immobilization to stabilize suspected spinal injury, the first step in treatment is prompt volume resuscitation.[73] If hemodynamic stability is achieved with volume alone, the use of vasoactive medications should be avoided. In the case of persistent neurogenic shock despite adequate volume resuscitation, prompt vasopressor support is required. We advocate that the initial vasopressor selected should have mixed alpha and beta activity, such as dopamine. This allows beta stimulation of the heart to treat bradycardia, and, at higher doses, provides alpha stimulation to counteract peripheral vasodilation.[74] Pure alpha agonists, such as phenylephrine, may be given to patients refractory to volume and dopamine. Corticosteroid use in spinal cord injury is highly controversial and is facility- and physician-dependent, but has no role in the immediate treatment of hypoperfusion from the loss of sympathetic stimulation.

Distributive/Septic Shock

Distributive and septic shock are the result of the body's innate response to infection and inflammation. The term distributive describes the fluid shift from the intravascular space to the interstitial space that accompanies this type of shock. Distributive shock rarely is seen in the acute trauma setting, although patients who have a delayed presentation following injury are susceptible. During the host response to infection, cellular mediated signals from infection-fighting neurotrophils and macrophages cause a wide array of clinical findings. Increased cardiac output (hyper-dynamic), peripheral vasodilation (edema), fever, and leukocytosis are all common findings.[75] In distributive shock, the first priority is volume resuscitation, followed by vasoactive support as needed. Prompt broad-spectrum antibiotic coverage should be given to those patients presenting with obvious delayed injury with infectious consequences.[76]

End Points of Resuscitation

The resuscitative phase of care in traumatic shock often is ongoing upon patient transfer from the ED to the ICU, operating room, or receiving facility. However, ED physicians and nurses need to be familiar with common end points of resuscitation. These are measures of resuscitative success, and they help identify patients with occult hypoperfusion despite normalization of clinical vital signs. Commonly accepted endpoints include urine output, lactate, base deficit, and tissue pH measures.

Urine output is a non-invasive end point of resuscitation that is easily monitored in the emergency department with a urinary catheter. For the adult trauma patient, 0.5-1 cc/kg of urine per hour is accepted as adequate output, and less may indicate occult hypoperfusion in the patient with seemingly normal vital signs. Urine output must be interpreted with care as advanced age, pre-existing renal disease, medications, and many other factors affect urine output. Lactate is a byproduct of anaerobic metabolism, which occurs during tissue hypoperfusion. It can be used as an indirect measure of systemic oxygen debt and is available from most hospital laboratories. Excessive alcohol consumption, cocaine abuse, and ketoacidosis are just some of the many factors that can alter lactate levels and complicate interpretation. Several studies have disputed the relationship between lactate level and success of resuscitation.[77-79] Lactate elevation may be an imperfect indicator of hypoperfusion, but multiple studies have shown it to be an independent predictor of mortality and ICU length of stay.[80]

Arterial base deficit can be used as an estimation of oxygen debt in traumatic shock, as it estimates the degree of metabolic acidosis. Kincaid et al[81] correlated the failure of base deficit normalization with ongoing blood loss, multisystem organ failure, and increased mortality. As with lactate levels, base deficit measures are subject to a myriad of variables and must be interpreted cautiously. Direct measurement of tissue and cellular oxygenation in theory provides a direct measure of tissue oxygen debt, but in the past it has been cumbersome and limited to research environments. Improving technology is bringing these types of measurements to the bedside, and their clinical use and impact on mortality have yet to be studied on a large scale. New, non-invasive techniques to estimate tissue perfusion including gastric tonometry, near-infrared spectroscopy (NIRS), and sublingual capnometry may prove valuable in the trauma patient population. To date, there is not a single perfect end point of resuscitation, but several, when used with a patient's clinical picture, can help guide resuscitative therapy.

Rationale for Trauma Transfer

Several reasons exist for a decision to move a trauma patient to a different hospital. Some transfers are done after the resuscitative phase has ended. These typically are patients who require some sort of urgent but not emergent treatment not available at the initial hospital. Examples include the patient transferred from a Level 2 trauma center because of the need for an orthopedic trauma surgeon experienced in the care of acetabular or pilon fractures. Sometimes the most critical decision during the resuscitative phase of care is the identification of a need to transfer to a higher level of care.[82]

When the emergency physician, with or without collaboration with a local surgeon, determines that the patient care needs outweigh local resources, a decision to transfer must be made promptly. Examples of patients requiring emergency transfer may include:

- positive FAST scan without a surgeon available;
- Glasgow Coma Score deterioration without availability of a neurosurgeon;
- inability to perform a CT scan in a patient with closed head injury;
- significant burns if the transferring hospital is not a burn center[83];
- a significantly injured child if the transferring hospital lacks pediatric trauma care capabilities.[84]

Non-therapeutic testing should be avoided once a decision to transfer has been made. The sending physician should recognize the need for transfer as soon as possible. Sometimes, this decision is made during the primary survey and communicated as soon as it is safe to do so. The trauma team may recognize the need for outbound transfer while it continues to provide life-saving interventions. Quick, concise verbal communication to another trauma team member should be all that is required to initiate outbound trauma transfer. Physician-to-physician communication is vital to allow an appropriate activation decision to be made at the receiving hospital. This can occur immediately at the termination of the primary survey, assuming that another qualified examiner is capable of continuing the evaluation.

Summary

Shock in the trauma patient is a treatable condition and most commonly is the result of hemorrhage. Trauma system protocol and staff training and preparedness are vital for efficient management of traumatic shock patients. Resuscitation strategies continue to evolve as our understanding of the cellular effects of shock improves. End points of resuscitation should be incorporated into routine trauma care to prevent occult hypoperfusion and provide aggressive, adequate treatment to the most severely injured patients.

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Physician CME Questions

1. The leading cause of injury-related death in the United States is:
   A. gunshot wounds.
   B. motor-vehicle trauma, with most fatal crashes in urban settings.
   C. motor-vehicle trauma, with most fatal crashes in rural settings.
   D. falls.

2. The American College of Surgeons mandates that all but which of the following types of patients should undergo the highest-level trauma team activation?
   A. GCS < 8 with mechanism attributed to trauma
   B. Motor-vehicle collisions with ejection of the patient
   C. Emergency physician discretion
   D. Confirmed blood pressure < 90 at any time in adults and age-specific hypotension in children

3. For a trauma patient to be in shock, he or she must:
   A. have an arterial base deficit of (-20.)
   B. have a systolic blood pressure of less than 90 mmHg.
   C. have inadequate tissue perfusion.
   D. have urine output < 15 cc per hour.

4. If hemorrhage results in hypotension in a trauma patient, then:
   A. the patient should bypass the closest hospital and go directly to a Level I trauma center.
   B. the probability of death is higher than 50%.
   C. uncrossmatched blood should be administered until the blood pressure rises.
   D. MAST garments should be applied.

5. Class II hemorrhagic shock means that:
   A. the blood pressure is normal and the pulse pressure is decreased.
   B. the blood pressure is decreased and the pulse pressure is normal.
   C. more than 30% of the blood volume has been lost.
   D. tachycardia exists (HR > 120).

6. Focused Assessment with Sonography for Trauma:
   A. requires a radiologist or a surgeon to perform.
   B. screens for hemoperitoneum and hemopericardium.
   C. should always be followed by computerized tomography (CT).
   D. has sensitivity below 85% when performed by individuals formally trained in its use.

7. Transient responders:
   A. require emergent intervention (surgery, angioembolization, etc.).
   B. can be stabilized with additional crystalloid and blood.
   C. should undergo resuscitative thoracotomy.
   D. have blood in the pericardium and should have thoracentesis.

8. Beck's triad:
   A. refers to the anatomic zones in the neck where operative intervention for stab wounds is mandatory.
   B. refers to tension pneumothorax.
   C. refers to pericardial tamponade.
   D. identifies the proper zone for placement of diagnostic peritoneal lavage catheters.

9. Serum lactate:
   A. is a byproduct of aerobic metabolism, and elevations mean that hyperventilation has occurred.
   B. is exclusively the result of alcohol consumption, cocaine abuse, or ketoacidosis.
   C. is a direct method to measure tissue and cellular oxygenation.
   D. is an indirect measure of systemic oxygen debt.

10. If interhospital transfer (to a higher level of care) is being contemplated for an acutely injured patient from a hospital without a surgeon:
   A. physician-to-physician communication is vital to allow the receiving hospital to be best prepared to handle the incoming patient.
   B. computerized tomography (CT) should always be done to exclude intra-abdominal hemorrhage, a common cause of exsanguination en route.
   C. the transfer should wait until the x-rays are copied because this will save time for the receiving team.
   D. the sending hospital's emergency physician should always accompany the patient, even if the emergency department will be left without a physician for a short period of time.

CME Answer Key

1. C; 2. B; 3. C; 4. B; 5. A; 6. B; 7. A; 8. C; 9. D; 10. A

Emergency Medicine Reports CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

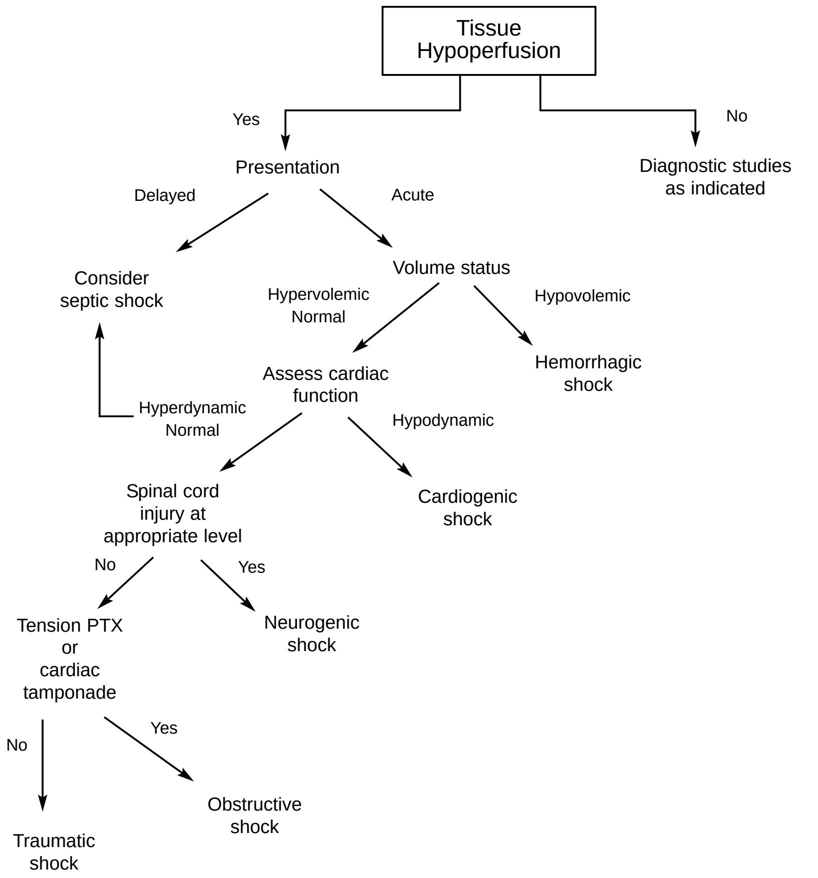
CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to evaluate their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. *After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Shock in the Trauma Patient

Tissue Hypoperfusion Algorithm



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Classes of Hemorrhage (70 kg Patient)

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood Loss (cc)	0-750	750-1500	1500-2000	> 2000
% of Blood Vol	0-15%	15-30%	30-40%	> 40%
Heart Rate	60-100	100-120	> 120	> 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Decreased	Decreased	Decreased
Mental Status	Anxious	Anxious	Anxious/confused	Confused/lethargic
Fluid Replacement	Crystalloid	Crystalloid	Crystalloid/blood	Crystalloid/blood

Common Indicators of Shock

PHYSICAL FINDINGS

- Decreased LOC/combativeness/stupor
- Cool, "clammy" skin
- Diminished pulses
- Capillary refill > 2 seconds
- Poor urine output

OBJECTIVE VITAL SIGNS

- Tachycardia
- Hypotension
- Tachypnea
- Hypothermia

METABOLIC MARKERS

- Elevated base deficit
- Decreased pH
- Elevated lactic acid
- Low bicarbonate

Intravenous Access Generalizations

- Peripheral access first
- Use the uninjured upper extremity first
- Use short, large-diameter catheters
- Use large-diameter drip set (blood tubing)
- Advanced access (central, "cut-down," intraosseous, etc.) is placed as the clinical circumstance and physician familiarity dictate
- Pressure infusing equipment should be immediately available
- First lab sent: Type and cross-match

Sites of Common Blood Loss with Associated Volumes

SITE	BLOOD LOSS (CC)
Thorax	> 5,000
Abdomen	> 3,000
Pelvis	> 2,000
Femur fracture	1,000-1,500
Humerus fracture	200-500
Tibia/fibula fracture	400-700

Supplement to *Emergency Medicine Reports*, January 1, 2009: "Approaching Shock in the Trauma Patient." *Authors:* **Marco J. Bonta, MD, FACS**, Medical Director, Trauma & Surgical Services, Riverside Methodist Hospital, Columbus, OH, Clinical Assistant Professor of Surgery, The Ohio State University, Columbus; and **Jonathan M. Enlow, DO**, Senior Surgical Resident, Riverside Methodist Hospital, Columbus, OH.